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Cardiomyopathy and heart failure secondary to anabolic-androgen steroid abuse

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ABSTRACT

Bodybuilders often use anabolic-androgenic steroids to improve performance. We report a case of a 30-year-old male bodybuilder with anabolic-androgen steroid abuse while getting ready for a bodybuilding contest. He had New York Heart Association class IV heart failure, severe nonischemic dilated cardiomyopathy, new-onset atrial fibrillation, cardiogenic pulmonary edema, and acute respiratory distress requiring mechanical ventilation. After 6 months of heart failure guideline–directed medical therapy, cessation of anabolic steroids, and maintenance of sinus rhythm, his ejection fraction improved.

KEYWORDS Anabolic-androgen steroid; guideline-directed medical therapy; heart failure; nonischemic cardiomyopathy

Anabolic-androgenic steroids (AAS) are commonly used by bodybuilders to enhance muscle mass. AAS contain supraphysiologic doses of testosterone and its analogues such as testosterone enanthate, nandrolone, and androstenedione. Supraphysiological doses and high-frequency usage of AAS have been linked to adverse cardiovascular effects.[1]

CASE PRESENTATION

A 31-year-old male bodybuilder with no prior medical history presented with a 3-day history of chest pressure, shortness of breath, hemoptysis, and palpitations. On examination, he had jugular venous distension, bilateral gynecomastia, lung rales, and a grade II/VI apical systolic murmur but no testicular atrophy or acne. He weighed 96 kg on presentation. Over the previous 6 months, he had been preparing for a bodybuilding contest and used testosterone enanthate 500 mg weekly. Three weeks prior to presentation, he increased his weekly dose to 1000 mg. His family history was positive for cardiomyopathy and heart failure in his mother (at age 49), for which she required an implantable cardioverter defibrillator.

An electrocardiogram *(Figure 1a)* showed atrial flutter with rapid ventricular response with 2:1 atrioventricular conduction. His heart rate was 130 to 150 beats/min, and his blood pressure was 109/64 mm Hg. He received intravenous adenosine 18 mg and a diltiazem bolus. He developed acute respiratory distress and was intubated and mechanically ventilated. Computed tomography of the chest showed bilateral interstitial pulmonary edema with pleural effusions. Transesophageal echocardiogram on day 2 showed severe left ventricular (LV) dysfunction with a left ventricular ejection fraction (LVEF) of 15%, mitral regurgitation, biatrial enlargement, and no left atrial appendage thrombus. He underwent direct current cardioversion with restoration of sinus rhythm; however, atrial flutter recurred within 24 hours. Pharmacologic therapy to maintain sinus rhythm included intravenous amiodarone followed by oral amiodarone.

His hemoglobin was 15.1 mg/dL; hematocrit, 46.1%; troponin, 0.1 ng/mL; and creatinine, 1.14 mg/dL. Secondary causes of cardiomyopathy were ruled out. SARS-CoV-2 antibody testing was negative. He was extubated 3 days later, and an echocardiogram on day 5 *(Figure 1b)* showed a moderately dilated LV size with an LV end diastolic dimension of 62 mm, severely reduced LVEF of 30%, global LV hypokinesis, and mitral regurgitation. Cardiac magnetic resonance imaging showed global LV hypokinesis with an LVEF of

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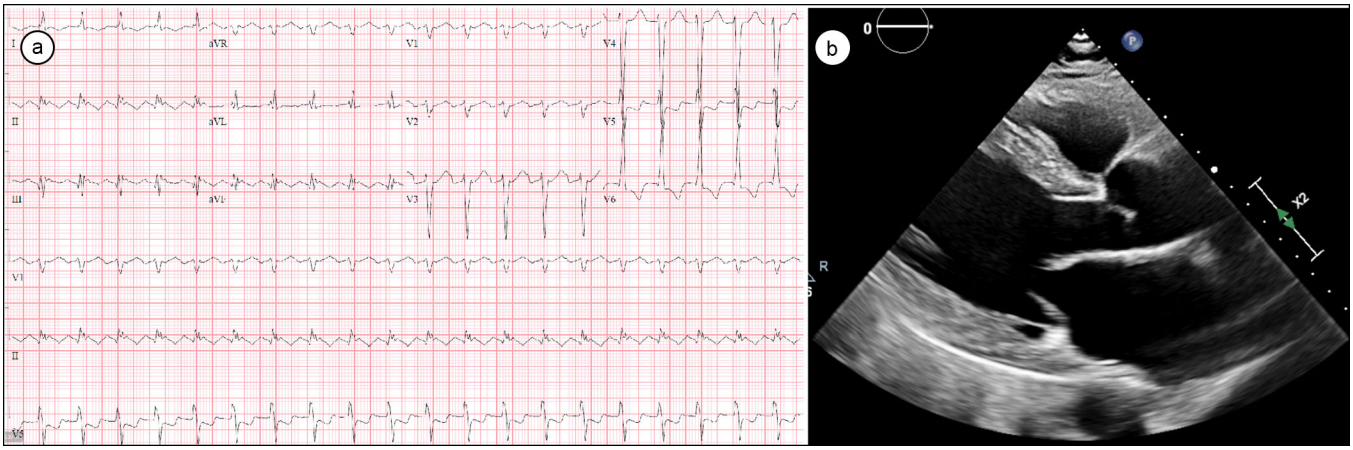

Figure 1. **(a)** Electrocardiogram showing atrial flutter with 2:1 atrioventricular conduction. **(b)** Echocardiogram, parasternal long axis view, showing moderately dilated left ventricle and left atrium.

Table 1. Cases of dilated cardiomyopathy in male bodybuilders using anabolic-androgen steroids

First author, year	Age (years)	LVEF (%)	LVEDD (mm)	AAS use (years)	Clinical presentation in addition to HF	LVEF later
Doleeb, 2019[10]	53	15	69	3	Dyspnea, palpitations, headache, syncope	6 months: 53%
Garner, 2018[6]	60	25–30	–	–	S3, S4, oliguria, pulmonary edema, pleural effusion, cardiomegaly	6 months: 50%–55%
Ha, 2018[7]	73	35	–	20	Tachycardia	—
Han, 2015[4]	30	15	–	7	—	2 years: 63%
Shamloul, 2014[11]	37	13	–	2	Elevated ALT, AST, INR	2 weeks: 20%
Youssef, 2011[12]	39	35	69	3	Dizziness, expressive aphasia, elevated CK	3 months: 40%–45%
Bispo, 2009[13]	40	15	–	10	RUQ pain; jaundice; elevated ALT, AST, INR, bilirubin, LDH; hepatomegaly	4 days: 25%
Ahlgrim, 2009[14]	41	18	67	4	CI 1.1 L/min/m^2, PCWP 40 mm Hg	Transplant evaluation

AAS indicates anabolic-androgen steroids; ALT, alanine transaminase; AST, aspartate transaminase; CI, cardiac index; CK, creatine kinase; HF, heart failure; IABP, intra-aortic balloon pump; INR, international normalized ratio; LDH, lactate dehydrogenase; LVEDD, left ventricular end diastolic diameter; LVEF, left ventricular ejection fraction; PCWP, pulmonary capillary wedge pressure, RUQ, right upper quadrant; –, no information available.

22% and a moderately dilated LV. Ischemic evaluation was deferred due to the low pretest probability of coronary artery disease.

His medication regimen included quadruple heart failure guideline–directed medical therapy: an angiotensin-converting enzyme inhibitor, lisinopril 5 mg daily; a beta-blocker, metoprolol succinate 50 mg daily; a mineralocorticoid receptor antagonist, spironolactone 25 mg daily; and a sodium glucose co-transport 2 inhibitor, empagliflozin 10 mg daily. He was on amiodarone and anticoagulated with apixaban for atrial flutter. Testosterone supplementation was initiated to prevent rebound low serum testosterone. Six months later, he was New York Heart Association class I, and a repeat echocardiogram showed an improved LVEF of 35% (moderate LV dysfunction). The risks and benefits of an implantable cardioverter defibrillator were discussed with the patient, and he opted to continue guideline-directed medical therapy and to proceed with device implantation if his LV function remained depressed.

DISCUSSION

Our case highlights AAS abuse as a cause of nonischemic cardiomyopathy, heart failure, atrial flutter, and acute cardiogenic pulmonary edema. The differential diagnosis included familial cardiomyopathy (absence of three generational family history) or AAS-induced cardiomyopathy. He was treated with quadruple heart failure guideline-directed medical therapy and cessation of AAS and achieved clinical stabilization despite his initial presentation. If diagnosed in the early stages, most cases of AAS-induced advanced heart failure are reversible, with full recovery of ventricular function. Low-dose testosterone supplementation to avoid rebound low testosterone is the mainstay of therapy.

Chronic resistance training may cause structural changes such as increased LV wall thickness and mass.[2] AAS increases production of reactive oxidative species, activates apoptotic pathways, and increases the activation of the renin-angiotensin-aldosterone system.[3] AAS has anabolic and androgenic properties that are cardiotoxic at supraphysiologic doses. Cardiovascular effects of AAS include LV hypertrophy, accelerated coronary atherosclerosis, a lower arrhythmia threshold, cardiomyopathy, and sudden death[1,4,5] and are due to fibrosis mediated by aldosterone-like and growth-promoting effects on the cardiac muscle. AAS promote increased viscosity through inducing a hypercoagulable state via increased platelet thromboxane A_2 receptor with concomitant polycythemia.

Cases of AAS-associated cardiomyopathy are usually seen in young men and are reversible with AAS cessation *(Table 1)*. However, cases of AAS cardiomyopathy are seen in older patients[6,7] with hemodynamic catastrophe requiring intra-aortic balloon pump and inotropes,[6] complicated by ventricular tachycardia,[7] and requiring an LV assist device.[8] In a retrospective review of 9 AAS cardiomyopathy patients, 30% (median age 31) required LV assist device implantation or were listed for heart transplantation.[9]

Clinicians need to consider androgen steroid abuse–associated nonischemic cardiomyopathy in young bodybuilders who present with de novo heart failure and cardiomyopathy without any other attributable risk factors.

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